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Reduced marine survival of hatchery-reared Atlantic salmon post-smolts exposed to aluminium and moderate acidification in freshwater

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ABSTRACT

Short-term Al-exposure and moderate acidification increased initial marine mortality in migrating post-smolts, and can thereby reduce viability of Atlantic salmon stocks. The delayed impact of short-term aluminium (Al) exposure on hatchery-reared Atlantic salmon smolt in moderately acidified freshwater (pH 5.88–5.98) was investigated during the first 37 km of the marine migration. Smolts were tagged with acoustic tags and exposed to low (28.3 ± 4.6 μg l^{-1} labile Al, 90 h) or high (48.5 ± 6.4 μg l^{-1} labile Al, 90 or 48 h) Al concentrations within the hatchery. Thereafter their movements, together with a control group, were monitored throughout the marine fjord. Al-exposure resulted in increased gill-Al and compromised hypoosmoregulatory capacity, as shown by elevated mortality in laboratory seawater challenge tests and reduced Na^+, K^+-ATPase activity levels. Further, Al-exposure resulted in decreased plasma concentrations of growth hormone (GH), while the insulin-like growth factor (IGF-I) was unaffected. There was a significant mortality in the 90 h high-Al group during exposure, and those surviving until release died during the first 3.6 km of the marine migration. Physiological stress and mortality were not only a result of the Al-concentrations, but also dependent on exposure duration, as shown by results from the 48 h high-Al group. Elevated mortality was not recorded in freshwater or after entering the sea for this group, which highly contrasts to the 100% mortality in the 90 h high-Al group, despite both groups having similarly high gill-Al levels. The low-Al group showed a 20% higher mortality compared to the control group during the first 10 km of the marine migration, but during the next 28 km, mortality rates did not differ. Hence, post-smolts surviving the first 10 km subsequently showed no differences in mortality compared to controls. At least one third of the mortality in both the low-Al and control groups were due to predation by marine fishes, indicating that the proximate cause for elevated mortality due to Al-exposure may have been predation. Migration speeds over 3.6, 9.6 or 37.1 km from the release site was not affected by Al-exposure.

1. Introduction

The Atlantic salmon is primarily an anadromous species with spawning and juvenile development taking place in freshwater, followed by a rapid growing phase in seawater, after which the sexually maturing adults migrate back to their native spawning grounds (Thorstad et al., 2011a). Anadromy is seen as an adaptive strategy, with individuals using the best suited habitat during different stages of the life cycle to increase fitness (Lucas and Baras, 2001). However, the transition from freshwater to marine habitats is a challenging phase that involves high mortalities (20–65% mortality in estuaries and fjords; Hvidsten and Møkkelgjerd, 1987;

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Thorstad et al., 2007; Davidsen et al., 2009). Preparatory morphological, biochemical, physiological and behavioural changes, which take place during parr-smolt transformation (smoltification), are necessary to preadapt the fish for life in the saline marine environment, which involves both long migrations and exposure to novel prey and predator species (McCormick et al., 1998). The marine mortality is mainly driven by density-independent factors, and the number of outward migrating post-smolts is positively correlated with the number of returning spawners of a cohort (Jonsson et al., 1998; Milner et al., 2003). Hence, factors affecting mortality during the smolt and post-smolt stages will contribute in determining population abundance (Milner et al., 2003). With the continued decline in wild salmon populations (ICES, 2011), it is imperative that salmon smolts exhibit high survival rates during their seaward migration.

The smoltification process may be adversely affected by a variety of anthropogenic factors, including acidification and pollutants (McCormick et al., 1998; Monette and McCormick, 2008). Acidification has caused the loss or reduction of numerous Atlantic salmon populations on both sides of the North Atlantic (e.g. Hesthagen and Hansen, 1991; Watt et al., 2000), and many rivers are still severely affected by chronic or episodic acid water (Kroglund et al., 2008; McCormick et al., 2009). Increased mortality due to acidification may be related to both high levels of H^+ (reduced pH) and aluminium (Al). During acidification, Al is mobilised from the soil, leading to elevated concentrations in the water (Teien et al., 2004b). The solubility of Al increases with decreasing pH, causing increased concentrations of inorganic monomeric Al (Al_i), which is the most toxic form to fish (Gensemer and Playle, 1999). As such, apart from when acidification is severe enough to be directly toxic, toxicity may also occur due to Al_i accumulating on the gill tissue, which may disrupt ion regulation and impair respiration (Rosseland and Staurnes, 1994; Gensemer and Playle, 1999). Loss of seawater tolerance in smolts during acid and Al exposure may be mediated, in part, by negative impacts on the GH-IGF-I system (Monette et al., 2008).

As long as the smolts remain in freshwater, moderate Al-exposure due to acidification may not be detrimental to the fish. However, as elevated gill-Al content can cause reduced hypo-osmoregulatory ability, sub-lethal exposure effects in freshwater may become lethal in seawater, causing increased marine mortality (Staurnes et al., 1993, 1996; Kroglund and Finstad, 2003; Kroglund et al., 2007). Such cross-over effects where the stressor is applied in one environment but the effect does not emerge until the fish has entered the new environment, are difficult to demonstrate in nature because contamination levels and mortality of individual fish are difficult to follow over time and through different environments. Previous studies have mainly been based on laboratory experiments coupled to limited tagging-recapture data.

This study aimed to use recent advances in telemetry techniques to test the hypothesis that the survival and migratory behaviour of acid and aluminium impacted Atlantic salmon smolts (*Salmo salar*) differ during their migration into the marine environment. The delayed impact of Al-exposure in moderately acidified freshwater was investigated during the first 37 km of the marine migration in a combined field and laboratory study. Hatchery-reared smolts were tagged with acoustic tags and exposed to zero (control group), low or high Al concentrations within the hatchery. After release, movements of the fish were monitored throughout a fjord using stationary acoustic receiver arrays. The physiological impact of the exposure on smolts (separate sub-groups kept under controlled experimental conditions in the hatchery) was determined by assessing seawater tolerance, branchial Al concentrations and Na^+, K^+-ATPase activity, as well as plasma levels of chloride, growth hormone (GH) and insulin-like growth factor (IGF-I).

2. Materials and methods

2.1. Study area

The study was performed in the laboratory of the Statkraft Energy AS hatchery situated at the outlet of the River Eira, and in the Romsdalsfjord system in Norway, 62°40′N 8°10′E (Fig. 1, Finstad et al., 2005). The area is not affected by acidification (Anon, 2011). The River Eira is a soft and clear water river, with a pH 6.7, 1–2 mg Ca l^{-1}, total Al <20 μg l^{-1}, Acid neutralising capacity (ANC) >50 μeq l^{-1}, low ion concentration, low conductivity (≈2.4 mS m^{-1}) and a low total organic content (<0.6 mg C l^{-1}) (Table 1).

2.2. Salinity and temperature in the fjord

Salinity and temperature were recorded from the surface to 30 m depth on 6, 8, 11 and 14 May 2009 at all receiver positions at site 1, and at three positions at each of sites 2, 3 and 4 (Valeport Ltd. miniCTD, Devon, UK, Fig. 1). In the inner fjord (sites 1–3), there was a brackish surface layer, with salinity increasing with depth. At 0.4 m depth, the average salinity was 21.3 (range 4.1–28.6), with the lowest salinities close to the river mouth. At 1.0 m depth, average salinity was 26.9 (range 22.8–28.6). A salinity of 30 was reached at average 8.6 m depth (range 6.0–11.3). In the outer fjord (site 4), salinities were never below 30.5 at any depth. Water temperature at 0–3.0 m depth, where smolts usually live (Hedger et al., 2011; Thorstad et al., 2011b), varied between 6.4 and 11.4 °C at all sites.

2.3. Fish and acoustic transmitter tagging

First generation hatchery-reared Atlantic salmon of the local River Eira stock were used. On 7 April 2009, pre-smolts (age 2+) were transferred from the Statkraft Energy AS hatchery to an experimental facility at the same location, where they were housed in tanks supplied with flow-through river water. On 14–15 April, 135 pre-smolts were randomly selected and surgically implanted with individually coded acoustic transmitters. Transmitters used were made by VEMCO Ltd., Canada, model V9P-1L-69KHz-S256 (dimensions 9 × 39 mm; weight in air/water of 5.2/2.7 g), having a depth sensor range of 50 m. The fish were anaesthetised (2-phenoxy-ethanol EC No 204-589-7, SIGMA Chemical Co., USA, 0.5–1.0 ml l^{-1} water) and transmitters were surgically implanted in the body cavity according to methods described by Finstad et al. (2005). Mean body mass of the smolts was 167 g (range 97–283) and mean total length 259 mm (range 213–314). Body size did not differ among the treatment groups (one-way ANOVA, mass: $F_{2,132} = 1.09$, $P = 0.34$, length: $F_{2,132} = 1.03$, $P = 0.36$).

2.4. Acid/aluminium exposure

Tagged fish were randomly divided into three experimental groups: a control group, a group exposed to moderately acidic water and low Al levels, and a group exposed to moderately acidic water and high Al levels (45 fish in each group). Full details of the water quality characteristics for the three experimental groups, as well as the background River Eira water are given in Table 1. Each of the three experimental groups was divided into three replicate sub-groups ($n = 15$), which were released on 5, 7 and 9 May 2009, referred to as release 1, 2 and 3, respectively. This sequential release over five days was carried out to reduce the risk of acoustic tag signal collisions and detection loss caused by several fish simultaneously passing receivers. Prior to release, each sub-group was transferred to exposure tanks (details below), acclimated there for